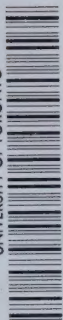


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PERIODONTAL STUDIES

Nos. 10, 11 and 12

by

HAROLD KEITH BOX, D.D.S., Ph.D.,

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University of Toronto

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STUDY NO. 10

CASE-MANAGEMENT: TREATMENT OF
SIMPLEX AND COMPLEX PERIODONTITIS



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STUDY NO. 10

CASE-MANAGEMENT: TREATMENT OF SIMPLEX AND COMPLEX PERIODONTITIS

- Outline - A. (a) Simplex Periodontitis; example of older type of conservative management
- (b) Other procedures for the elimination of pockets
- (c) Conservative type of management modified by the packing procedure
- (d) Conservative type, with packing, further supplemented by insufflation
- B. (a) Complex Periodontitis: example of older type of conservative management
- (b) Example of conservative type of management modified by the Dunlop supplements
- C. Treatment of the Pocket
- D. Vitamin Therapy
- E. Bibliography

CASE MANAGEMENT: TREATMENT OF SIMPLEX AND COMPLEX PERIODONTITIS

A. (a) Simplex Periodontitis: example of older type of conservative management.

1. Superficial scaling, and polishing of crowns
2. Extraction of hopelessly diseased teeth
3. Correction of faulty contact points
4. Deep scaling - combined with cemental and periodontal curettement where reattachment is the aim of the operator
5. Thorough instruction in the care of the mouth

(b) The other procedures in common use for the elimination of pockets are three in number, viz:

1. Radical surgery, e.g: the techniques advocated by Black, Ward and others
2. Electrosurgical method of Webb
3. Conservative surgery, e.g: the surgical flap and semiflap technique of Kirkland

(c) Conservative type of case-management modified by the packing procedure.

Example -

Sitting one

(1) Superficial scaling: the scalers are dipped into a mixture comprised of the following components:

Mentho-Borate paste - 1/4 teaspoon
 ABM mixture - approximately 5 grains on tip of spatula
 Oil of peppermint - 1 drop
 Alcohol - 4 drops

Also, some of this mixture is usually carried to the gingival crevices and proximal tissues, if deposits are abundant, in advance of scaling, and allowed to lie there and permeate the interstices of these regions. As the term superficial scaling implies that the deposits are mainly supragingival, the removal of these deposits with a minimum of gingival disturbance is to be advised at this stage. Crushing or laceration of the soft tissues is to be strictly avoided.

(2) Cleansing and polishing of the clinical crowns: This is carried out with Polishing Mixture No. 1 and the rubber cup

(3) Paste Suffusion: The crevices, pockets and embrasures are thoroughly flushed with Mentho-Borate paste.

(4) Paraffin Packing: The interproximal spaces are thoroughly packed, immediately following the use of the paste.

Sitting two (two days later)

(1) Further scaling where calculus is visible. No attempt is made at this time to remove the calculus from the pocket depths.

(2) Paste Suffusion and Paraffin Packing: The packing is forced deeper into the crevices and pockets, and inserted again interproximally.

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(2) Paste Suffusion and Paraffin Packing: The packing is forced deeper into the crevices and pockets, and inserted again interproximally.

Sitting three (two or three days later)

(1) Deeper scaling can now be carried out as a rule with little injury to the soft tissues. This method offers this manifest advantage over the older type of conservative management. Through reparative changes in the tissues generally and on the pocket wall in particular, and because the deposits of calculus are rendered visible and more accessible, the danger of tissue traumatization and forcing of microorganisms into the blood-stream is reduced to a minimum. The procedures of flushing the crevices and pockets with paste, and packing with paraffin prior to the removal of deeper deposits of calculus, are in harmony with the newer knowledge of gingival pathology.

(2) Polishing with Mixture No. 2

(3) Paste Suffusion and Paraffin Packing

(4) Instruction in the care of the mouth for sanitation and gingival stimulation carried out as before.

(d) Conservative type of management, with packing, and further supplemented by the insufflation procedure.

As a rule, insufflation with Dunlop vapor may be begun at the second or third sitting. It must be remembered that it is good practice to endeavor to reach tissues showing marked involvement from insufflation sites in tissues that appear relatively sound.

B. (a) Complex Periodontitis: example of older type of conservative management.

- (1) Superficial scaling of teeth indicated for extraction
- (2) Extraction of hopelessly diseased teeth
- (3) Establishment of the most ideal conditions of occlusal function possible (occlusal equilibration)
- (4) Superficial scaling: cleansing and polishing of the clinical crowns
- (5) Treatment of the pocket - combination of deep scaling, cemental and periodontal curettement where reattachment is attempted
- (6) Instruction in the care of the mouth

That the extraction of condemned teeth should precede the adjustment of occlusion is quite apparent. Where no teeth are to be removed, it is to be noted that the correction of occlusal and articular conditions is the first step in this type of case-management of Complex Periodontitis.

The writer¹ has pointed out in a former treatise that any procedure which changes the gingival picture in any detail prior to the correction of traumatogenic occlusion and articulation is strictly contraindicated. This of course is because the true underlying conditions would be masked. In this class belong the procedures of scaling, polishing, packing, the application of medicaments, pastes or salves, and the use of vaccines, astringent or antiseptic mouthwashes. In a brief paper² twenty years ago, the writer called attention to this obscuring influence of scaling on the gingival signs of traumatogenic occlusion. Further, it has been emphasized that nothing be done to change the patient's habits of oral and dental care and it has been stated that it is best to avoid at this time any discussion of the subject with the patient. The institution of

a correct method of using the tooth brush, for example, would undoubtedly serve to disturb the finer details of the gingival picture.

The writer still thinks that, as a general rule, it is not advisable for the beginner in periodontia to do anything which will obscure certain gingival signs caused by or related to traumatogenic occlusion. It is of considerable importance to be able to recognize in the gingival tissues the effects of pericemental overstress and the changes which take place in the tissues when, of the etiological factors, it alone has been removed.

When the operator has become more expert in his management of occlusion and articulation, the changing of the gingival picture prior to the adjustment of occlusion is of less consequence. Experience has taught that the problem of the increased mobility of the tooth in its alveolus is of such significance that, as a rule, in Complex Periodontitis, it becomes the symptom of greatest interest. Hence, the early use of a supplementary procedure like the Dunlop insufflation, which in itself so markedly increases tooth stability, would seem to be based on sound therapeutics. In fact the partial stabilization of the teeth in their sockets prior to occlusal equilibration in most cases would appear to be an advantage.

(b) Complex Periodontitis: example of conservative type of management modified by the Dunlop supplements.

Sitting one

- (1) Superficial scaling (supragingival)
- (2) Cleansing and polishing of the clinical crowns; polishing mixtures Nos. 1 and 2
- (3) Insufflation of Dunlop vapor.

It was pointed out previously that the reduction of gingival edema is shown clinically by the transformation of the smooth and shiny surface into one of "stippling". Also the volume of the tissues is reduced with the return of normal contour. The atrophied epithelium tends to be restored to normal, and bleeding is greatly lessened or disappears altogether. Reduction of the gingival edema is to be noted almost invariably on the second sitting when the above procedures have been carried out.

It is recognized that phagocytosis is the main defense of the gingival tissues against infection by serophytic bacteria, that is by bacteria like the staphylococcus, streptococcus and pneumococcus (micro-organisms that proliferate in the normal serum). These bacteria are engulfed by the leucocytes after they have been acted upon by the blood serum and as stated in an earlier paper³ "the leucocytes produce anti-bacterial substances which have the power of killing ingested living bacteria, and when this is accomplished the microbes are digested by intracellular enzymes."

Arkin⁴ showed that agents facilitating oxidation favor the phagocytosis of bacteria by leucocytes. Findlay⁵ has pointed out that anti-septics have an inhibitory action on phagocytosis, and Fleming⁶ has shown that they decrease the bactericidal power of the leucocytes.

The use of hypertonic salt solutions as a mouthwash has been advocated by the writer,⁷ in the therapy of mild though frequently tenacious

suppurative inflammations of the gingival tissues. These solutions "besides washing from the crevices food debris, bacteria and leucocytes, tend to establish for a time through osmosis, a flow of fluid from the tissues into the crevice. This lymphagogue effect is not only of value in drawing to the infective focus fresh lymph containing opsonins, etc. but also directly attracts a supply of undamaged leucocytes to the focus. In this way the natural defensive mechanisms are brought into play and incidentally the deleterious effect of the leucocidin may be overcome."

Sitting two

- (1) Insufflation with Dunlop vapor
 - (2) Paste suffusion of crevices and pockets
 - (3) Packing of pockets,
- or
- (1) Insufflation with Dunlop vapor
 - (2) Establishment of the most ideal conditions of occlusal function possible
 - (3) Paste suffusion and paraffin packing

The object of the adjustment of occlusal and articular relationships is primarily the establishment of heavy function on each tooth. Insofar as this is achieved with a favorable type of loading will the periodontal membrane and supporting bone tend to become stronger and well developed. In dealing with individual problems of loose teeth, special consideration is given to the mechanics of the opposing dento-periodontal units. This may be regarded as a specific remedial procedure.

Classification of abnormal articulations commonly observed in practice

- i. Triangle types: (1) within the "zone of tolerance"
- (2) outside the "zone of tolerance"

These terms relate to the distance of separation in the posterior molar regions, when the cuspids are in right and left lateral occlusions, i.e: resting contact relationships. If the greatest separation found in either right or left lateral occlusion is approximately 1 millimeter, this is said to be within the "zone of tolerance". Separations of more than 1 millimeter are, as a rule, considered to be outside this "zone of tolerance".

- ii. End-to-end types: (1) disturbance in centric occlusion
- (2) open-bite with posterior overloading
- iii. Vertical overbite types: (1) articulation is established in one or both strict lateral directions. (As a rule periodontal disease is absent on the anterior teeth.)
- (2) no articulation is established. (Periodontal disease is frequently present about the anteriors.)

The procedures of occlusal adjustments and establishment of articulations are obviously best taught in the clinic. Nevertheless, the following brief description of the steps usually undertaken by the writer in the correction of class i(1), viz: the triangle type within the "zone of tolerance", may be of interest:

(1) Study of right and left triangles in the lateral positions; the distance of separation in the posterior molar regions is measured approximately in both right and left lateral positions with the cuspids in contact and generally in line with each other. The triangle with the widest separation is chosen as the one to be adjusted first.

(2) Adjustments now are made to bring this triangle into cuspid-molar-molar contact. This is an occlusion, in other words, a resting contact relationship. Let us assume, in this case, that this triangle is the right lateral one.

(3) Next, adjustments are made in the left lateral position to bring the left triangle into cuspid-molar-molar contact. It is frequently to be observed that correction of the first triangle greatly improves the second triangle as to contacts. And, it is hardly necessary to add that in establishing the second triangle, it is important to avoid grinding occlusal areas that belong to the first established triangle. To prevent confusion in this regard Dr. W. Y. Hayden of London, Ontario, has suggested the use of two colors of articulating paper.

(4) The anterior teeth in the protrusive occlusal position are now adjusted, care again being taken, if the cuspids are involved, to avoid interfering with areas involved in the two triangle relationships.

(5) The corrections in centric occlusion now are made. Here once more it is essential not to interfere with the right and left lateral triangles while making the centric adjustments. The use of red and blue articulating papers is decidedly an advantage here.

(6) As the articulations are based on mandibular movements from the eccentric positions towards the centric, the next move is an endeavor to establish the sliding articulations. When the first adjustments have been carefully carried out, in class I (1), this is frequently a comparatively simple matter. To ensure elimination of minor disturbances, and to more thoroughly and accurately establish the natural plane of wear, the use of an abrasive adjunct as introduced by Linghorne⁸ is of distinct value. As pointed out previously, its use should be under the observation and control of the dentist. This natural plane of wear, in the writer's opinion, is dependent on a number of factors and should tend to be produced, in the first and second molar, and bicuspid regions, at the expense of the upper lingual and the lower buccal cusps.

Sitting three (and subsequent sittings dependent on the type of case and extent of the disease) may comprise the following procedures:

- (1) Subgingival scaling
- (2) Paste suffusion
- (3) Paraffin packing

or

- (1) Insufflation with Dunlop vapor
- (2) Paste suffusion
- (3) Paraffin packing
- (4) Instruction in the care of the mouth

It should be pointed out that it is not sound practice to undertake subgingival scaling and insufflation at the same sitting. Scaling, polishing, paste suffusion and paraffin packing make a satisfactory combination. It is not the practice of the writer to treat the pockets until a widespread sanative influence has been manifested in the periodontal tissues. This is requisite before treatment of the pockets is carried out.

C. Treatment of the Pocket

The radical and conservative surgical methods of pocket elimination in common practice have been referred to previously. The reader is advised to consult some of the more recent text-books^{9 10} for further information on these techniques. A conservative method suitable in certain types of pockets has been thoroughly described by the writer.¹¹ The aim of this method is the reattachment of the soft tissues to the root. The procedures to be undertaken comprise the following:

- (1) Scaling
- (2) Cemental curettement
- (3) Periodontal curettement
- (4) Establishment of the clot
- (5) Sealing of the pocket

The absolute test for fibrous reattachment in treated cases of periodontal pockets is a shift of the pocket base toward the enamel line. If during the surgical treatment of the pocket, the tissues beneath the pocket base have been severed from the cementum, reattachment of these tissues to the cementum may take place. It is plain that this reattachment will not move the pocket base crownward. This occurrence, and the atrophy of the gingival margin have been given as arguments to explain why clinicians have been mistaken in their belief in a true reattachment of the soft tissue wall to the cementum.

Obviously then, in proving reattachment of the soft tissues, the pocket base must be measured from some fixed mark on the tooth itself, and measured before and after the carrying out of all the procedures designed to promote reattachment.

It has been shown by experiment that the predominating factor controlling reattachment of the soft tissues through the medium of cementum deposition is the local health of the tissues at the time of the operation. If healthy soft tissues easily attach themselves to the cementum, and unhealthy tissues do not, it is apparent that it would be folly to expect reattachment without first devoting considerable attention to the promotion of healthy states and reparative processes in the soft tissues. It is hardly conceivable that any dentist would expect reattachment following the employment of the reattachment technique at the first sitting, and in chronically inflamed edematous soft tissues. Only when the tissues are brought to a healthy state comparable to healthy tissues in control areas of the mouth should any reattachment technique be undertaken. This is one of the prerequisite aims of the case-management previously outlined.

The treatment of choice in the majority of periodontal pockets in the writer's practice is the method of paraffin packing combined, when indicated, with some gingival resection. Very fine results may be achieved by repeated paraffin packings, removal of deposits and freshening of the cemental surface. The reduction in height of the soft tissue wall, combined with an apparent gradual shifting of the pocket base toward the enamel line during the reparative process, contributes greatly to the shallowing of the pocket. And, where the pockets are deep and isolated, the excision of a small wedge-shaped piece of tissue from the wall of the pocket, combined with paraffin packing, is most effective.

The writer stated in a previous paper¹² that the production and maintenance of a cornified crevicular epithelium is the ideal end result. Gottlieb¹³ has suggested the use of tannic-acid containing drugs for cornification of the epithelium in the crevices. Recently, Mr. Fenton, our Research Technician, has suggested to the writer the use of a hitherto untried chemical substance, and at present it is being given a thorough trial in clinical practice. The results to date have been very satisfactory and the work is still under observation.

D. Vitamin Therapy

Sinclair,¹⁴ as referred to in a previous study, has stated that his observations suggest that complex periodontitis is the result of a multiple vitamin deficiency. In treating cases of periodontal disease, and Vincent's infection, he advocates the use of vitamin A and vitamin B complex as a "prophylactic precaution".

The following are Sinclair's recommendations:

- (1) Vitamin A deficiency - 50,000 international units of vitamin A for 30 days; then the dosage is reduced to 25,000 international units for 30 days or somewhat longer.
- (2) Vitamin B deficiencies - vitamin B and G (B₂) capsules; 4 to 6 capsules a day, supplying 600-900 international units of B₁, and 600-900 gammas of riboflavin (G or B₂), besides other factors of the B complex. This dosage is advised for 30 days, after which it is reduced to one-half for an indefinite time. In addition to vitamin B complex, Sinclair advocates for the more extensive cases, 100 mg. of nicotinic acid and 10 mg. of thiamin chloride for 10 days.
- (3) Vitamin C deficiency - 200 mg. of cevitic acid daily for 10 days, then 100 mg. per day as indicated.

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STUDY NO. 11

NECROTIC GINGIVITIS

STUDY NO. 11

NECROTIC GINGIVITIS

Outline - A. Definition

B. Synonyms

C. Signs and Symptoms

D. General Description of the Course of Necrotic Gingivitis

E. Etiology

(a) Bacterial Factor

(b) Oral Conditions Favoring Anaerobic Growth of Bacteria

F. Histo-pathology of Necrotic Gingivitis

G. Treatment

(a) Treatment of the Acute Phase

(b) Elimination of the "secondary incubation zones"

(c) Elimination of the "primary incubation zones"

H. Bibliography

NECROTIC GINGIVITIS*

A. Definition

Necrotic gingivitis is an infectious inflammation of the gingival tissues, exhibiting lesions characteristic as to location and nature and occurring only in mouths where teeth are present. It is characterized by a type of pseudomembrane formation--at least by a soft layer composed mainly of bacteria and necrotic tissue--and superficial ulceration. The disease frequently spreads widely and rapidly, involving other mucous membranes of the mouth, thus establishing a stomatitis as an added complication.

B. Synonyms

Trench Mouth
 Trench Gums
 Phagedenic Gingivitis
 Acute Ulcerous Gingivitis
 Acute Ulcerative Gingivitis
 Ulcerative Gingivitis
 Ulcerative Stomatitis
 Ulcero-membranous Gingivitis
 Ulcero-membranous Stomatitis
 Vincent's Gingivitis
 Vincent's Periodontitis
 Vincent's Stomatitis
 Vincent's Angina
 Vincent's Peridental Angina
 Plaut-Vincent's Stomatitis
 Stomatitis Ulcerosa
 Stomatitis Ulcero-membranacea
 Fusio-spirillary Gingivitis
 Fusio-spirillary Marginal Gingivitis
 Fusio-spirillary Peridental Gingivitis
 Fusio-spirillary Peridental Gingivitis
 Fusio-spirillary Periodontitis
 Foetid Stomatitis
 Putrid Stomatitis
 Putrid Sore Mouth
 Stomatocace
 Stomacace
 Acute Septic Gingivitis
 Pseudomembranous Angina
 Spirochaetal Stomatitis

*This study is comprised, in the main, of excerpts drawn from Bulletin No. 14 of the same name, and issued by the Canadian Dental Research Foundation in 1930.

C. Signs and Symptoms

(a) Of first importance in the syndrome is the presence on the gingival margin of a destructive lesion covered by a grayish, grayish-yellow, yellowish-white or greenish-white pasty slough or pellicle which when rubbed or lifted off leaves a sensitive bleeding surface. Preceding the destruction, the area affected is as a rule red, tumefied* and very sensitive. It tends to bleed readily and sometimes profusely after the slightest injury. This, however, is soon followed by the formation of a pseudomembrane-like deposit which at this stage has a great deal of resemblance to that caused by the Klebs-Loeffler bacillus and with which it is sometimes confused. Hardgrove¹ states that in Vincent's infection of the tonsil this pseudomembrane is not so white as that found in diphtheria and is more easily removed. It must be kept in mind, however, that in the more severe forms of infection with the diphtheria bacillus, the pseudomembrane may also be yellow, gray, olive-coloured, or where hemorrhages accompany the inflammatory process, more or less black. In necrotic gingivitis, the ulcers have a well-defined margin and clinically have often been wrongly diagnosed as syphilis. The tissue-destruction in its peculiar involvement of certain portions of the gingival contour gives the tissue a characteristic eroded, "punched-out" or "dissected away" appearance. Because the ulceration is of a rapidly and widely spreading nature, the term phagedenic gingivitis was applied to it by early writers on the subject (Coplin).² Years later, Stillman and McCall³ adopted the same appellation for this disease. Through involvement of the alveolar process and pericementum, a special type of simplex periodontitis⁴ is frequently established. From our present knowledge of these lesions, based on the pathological findings and in the absence of positive proof of the primary etiological factors, it is felt that the term "necrotic gingivitis" best designates this disease.

(b) Next in importance in the syndrome is the distinctive and peculiarly offensive odour. The breath becomes intensely foul, and this putrid, fetid odour once experienced is alone almost sufficiently characteristic to establish a diagnosis of necrotic gingivitis.

(c) Another symptom of major significance is the factor of sudden onset. Frequently the patient can state the date of the appearance of the main symptoms of the disease. This is particularly true in cases of epidemic type.

(d) The gingival tissues are usually very tender, and in some instances so painful as to interfere with normal mastication of food.

*A lesion involving the distal gingivae of lower third molars and the soft loosely attached tissues posterior to them and resembling in some respects the early tissue changes in necrotic gingivitis is extremely common in acute lymphatic leukemia and terminal exacerbations of chronic lymphatic leukemia. This is commonly spoken of as a lesion of leukemia. The tissues present a solid, heaped-up, turgescient and nodular appearance. Sir Kenneth Goadby, K.B.E., referring to this lesion in his book "Diseases of the Gums and Oral Mucous Membrane" states that there is no necrotic ulceration of the gum edges and that from the buccal sulcus upwards, the whole gum was affected.

(e) There is an increased flow of saliva, frequently bloody from slight gingival hemorrhages, and usually of a metallic taste.

(f) Frequently there is a feeling of uneasiness and mouth discomfort evidenced at times by a wedging sensation in the interproximal spaces.

(g) Patients sometimes state that the teeth feel "wooden" or "dead" to the bite, as if the pericementum were anaesthetized.

(h) Occasionally there is swelling and pitting of the tongue.

(i) The submaxillary glands and other neck glands may be swollen.

(j) The presence of macroscopic pus is not a requisite sign.

(k) Loosening of the teeth is not characteristic but may be found in the late stages of the disease.

(l) General symptoms such as malaise, increased temperature, accelerated pulse rate, pallor of the skin, mental depression, insomnia, vertigo, restlessness, nocturnal mental hyper-activity and lack of appetite are common.

(m) Gastric disturbances such as acidity, flatulence, regurgitation and occasional vomiting are also familiar symptoms.

(n) Alternating diarrhoea and constipation are not uncommon.

(o) An exanthematous eruption resembling measles is sometimes observed.

Many cases are observed in which the history gives evidence of a previous necrotic gingivitis. In these cases most of the foregoing symptoms are absent or are very much modified. The characteristic sign is the "punched-out" appearance of the labial or lingual aspects of the interproximal gingivae. In addition, the bulk of the interproximal gingiva is also still further cupped out, creating thereby a crater-like depression the sanitation of which is impossible by care on the part of the patient. Bleeding from these areas is common, although seldom as severe as in the acute stage. It is the opinion of many periodontists that this form of subacute or chronic gingivitis bears some etiologic relation to the acute or necrotic type. Indeed some investigators consider necrotic and chronic gingivitis to be two types of oral spirochetosis, the first an acute form, and the second a chronic form.

Doubleday⁵ has stated: "On what does the suggestion that there really is such a condition as chronic fusio-spirillary infection of the gum rest? I think one may put forward as good evidence the undoubted fact that there is a clinical condition of the gum characterized by the presence of serum and blood, but little or no pus, in which these organisms are found in direct smears, and that when in the course of treatment the organisms gradually disappear the clinical signs clear up as well."

Gangrenous Stomatitis.--This disease fortunately is a comparatively rare one and is usually observed in children from two to five years of age. It begins in unusually debilitated children, and as a rule during convalescence from infectious diseases such as scarlet fever, typhoid fever, diphtheria, and as Osler has pointed out, in particular, measles. The first lesion is a small nodule, dense and sensitive, appearing on the gum or inside of the cheek. A gangrenous process follows, attended by sloughing of the involved structures. In mild cases, the primary necrosis may be limited to one of the starting points and with healing may leave the parts deformed--and disfigured if penetration of the cheek has occurred. The disease advances by a progressive necrosis and, as a rule, this process extends rapidly, perforating the cheek and involving the chin, tongue, jaws and sometimes even the ears and eyelids. The soft parts are converted into blackish-green or brownish foul-smelling masses and the bone becomes necrotic. The disease usually terminates fatally in from one to two weeks by septicemic processes.

The histo-pathology of gangrenous stomatitis will be discussed later in its relation to the histo-pathology of necrotic gingivitis.

D. General Description of the Course of Necrotic Gingivitis

All cases of necrotic gingivitis and stomatitis may be divided into two classes, viz:

(a) Those in which the disease has been established by activation of the microorganisms through the presence in the mouth of all the necessary factors for rapid anaërobic growth.

(b) Those in which the disease has been established through contagion, i.e: the communication by mediate or immediate contact with virulent microorganisms.

Close observation has revealed that in the first class a rapid multiplication of the fusiform bacillus and incidentally the spirillum takes place in certain regions through a combination of conditions which favour anaërobic growth. These regions are termed by the writer "primary incubation zones". Through extremely favourable cultural conditions in these sites, the bacteria become more virulent. Fine examples of "primary incubation zones", in the order of their importance are as follows:

1. Gingival flaps on partly erupted lower third molars.
2. Palatal margins of interproximal gingivae of upper centrals associated with deep gingival crevices or pre-existing periodontal pockets.
3. Buccal gingival margins of molars in crowded contact with folds of the mucous membrane of the cheek.
4. The tonsils: (it must not be overlooked that the tonsils frequently furnish a good example of "primary incubation zones".)

It is well known that bacteria at any point on the upward curve of growth are more liable to spread and grow under conditions somewhat less favourable for their growth. In other words they become more virulent. This has been shown for pneumococci by Bloomfield and Felty⁶ and is

probably a general characteristic for all bacteria, including fusiform bacilli and Vincent's spirilla. This would account for their new colonization in relatively unfavourable sites, in which, however, due to certain depressing conditions, their multiplication is permitted. These sites have been termed by the writer "secondary incubation zones". Here rapid proliferation and resulting increased virulence may again take place, thus continuing the process, with the exhibition of the characteristic symptoms of the disease. Examples of "secondary incubation zones" are as follows:

(a) Gingival tissues of teeth in traumatogenic occlusion.

(b) Gingival tissues, having a pre-existing inflammation; calculus, rough margins of dental restorations, mouth breathing and excessive smoking are common causes. (Here, as a rule, we are dealing with a consecutive infection, that is to say, the Vincent's infection is implanted upon an infective process already established.)

(c) Periodontal pockets.

When the infection has been established through contagion, as in the second class, both the primary and secondary incubation zones may become the sites of the lesions. As stated previously, the virulence of the microorganisms in part determines their establishment in relatively unfavourable sites, as in the secondary incubation zones, and as Hardgrove has stated, the more virulent the microorganisms, the more likely they are to operate on a free surface. The free mucous surfaces of the gingivae, alveolar mucosa, cheeks, tongue, and floor of the mouth are obviously less favourable sites than the "secondary incubation zones", but in severe cases where the microorganisms are especially virulent, these frequently exhibit the characteristic lesions of necrotic gingivitis.

Examples of mediate contagion, i.e: contagion conveyed by a person or object from the sick to the well, are found in infection from common drinking vessels, eating utensils in restaurants, trains and steamships, also from door-knobs, telephone receivers, and common towels.* Immediate or direct contagion, i.e: contagion from direct contact with a sick person, is well exemplified in coughing, or kissing.

In many cases, the highly acute phase of necrotic gingivitis lasts from four to seven days; in others, however, the period is much longer and may last for several weeks. With diminution of the more severe symptoms, the acute phase is followed by one of subacute or chronic infection.

In these chronic areas, both primary and secondary, containing fusiform bacilli and spirilla, conditions allowing a very slow multiplication are apparently present. With favourable cultural conditions, the microorganisms may take on a rapid growth and virulence with exacerbation of the old process. That trauma may be a factor in this exacerbation is well known to the dental practitioner. As Hirschfeld⁷ has pointed out, scaling and extraction are frequently followed, in from twenty-four to thirty-six hours, by symptoms of so-called acute Vincent's infection.

*Sir Frank Colyer, on his last visit to Toronto called the author's attention to his interesting observation that necrotic gingivitis was frequently seen in hostlers.

E. Etiology

(a) Bacterial Factor

The nature of the lesions was not clear until Plaut,⁸ Vincent⁹ and others reported uniform bacteriological findings. In smears taken from the lesions, there is found a spindle-shaped or fusiform bacillus with which is associated as a rule a long spirillum, not unlike the spirillum of relapsing fever. Abel¹⁰ first succeeded in growing the *Bacillus fusiformis* in the laboratory. It was grown in mixed cultures with a contaminating Gram-negative coccus, and disappeared on sub-culture. Following numerous attempts of many investigators, it was finally grown in pure culture.

The relation of the two microorganisms, the bacillus and spirillum, has been the subject of much controversy. The work of Tunnicliff¹¹ favours the viewpoint that these two forms are different developmental phases of one organism. This investigator cultivated the organisms anaerobically upon slants of ascitic agar at 37.5°C. Before the fifth day, the fusiform bacilli only could be found but later the spirilla appeared. From the observation that finally the spirilla predominated, it appeared that they represented the adult form, having developed out of the bacilli.

On the other hand, these two microorganisms are regarded by other workers as two distinct species closely associated in symbiosis. Mühlens and Hartmann¹² in isolating from the same case both the spirilla and fusiform bacilli, found the colonies to be quite distinct. They were unable, further, to find evidence of the mutation of these organisms. Later, the findings of Krumwiede¹³ confirmed the observations of Mühlens and Hartmann.

Hiss and Zinsser¹⁴ have given the following description of these microorganisms: "The fusiform bacilli described by Vincent, Plaut and others are from 3 to 10 micra in length and have a thickness at the centre varying from 0.5 to 0.8 micron. From the centre they taper gradually toward the ends, ending in blunt or sharp points. The length of these bacilli may vary greatly within one and the same smear preparation. They are usually straight, sometimes slightly curved. They do not stain very easily with the weaker anilin dyes but are readily stained by Loeffler's methylene-blue, carbol fuchsin, or better by Giemsa's stain. Stained by Gram, they are usually decolorized though in this respect the writers have found them to vary. Stained preparations show a characteristic inequality in the intensity of the stain, the bacilli being more deeply stained near the end and showing a banded or striped alternation of stained and unstained areas in the central body. Their staining qualities in this respect are not unlike those of the diphtheria bacillus, and according to Babes¹⁵ the dark areas are to be interpreted as metachromatic granules. The bacilli are not motile."

The spirilla found in Vincent's angina are usually somewhat longer than the fusiform bacilli and are made up of a variable number of undulations, shallow and irregular in their curvatures, unlike the more regularly steep waves of *Spirochaete pallida*. They are stained with even more difficulty than the bacilli and usually appear less distinct in the preparations. The stain, however, is taken without irregularity showing none of the apparent metachromatism observed in the bacilli."

These microorganisms when found in smears taken from the lesions of necrotic gingivitis are occasionally present almost in pure culture. As a rule, however, they are accompanied by other oral bacteria which undoubtedly flourish under these conditions. Smears usually contain the material from the surface of the lesions, and as would be expected, the difficulty of obtaining pure smears of the *Bacillus fusiformis* and Vincent's spirillum in these mixed types, is almost insuperable. That these microorganisms are the primary etiological factors in these inflammations has never been demonstrated conclusively. Because of their common association with other bacteria, it has been pointed out that they may be secondary invaders upon a background prepared for them by other bacteria. "On the other hand," as Goadby¹⁶ has stated, "the curious mass of bacteria found present in ulcero-membranous stomatitis, the tissue necrosis which is everywhere in progress, and the large number of bacteria penetrating the tissue in all directions seem difficult to dissociate from some definite role in the disease."

(b) Oral Conditions Favouring Anaërobic Growth of Bacteria

1. Primary Incubation Zones

A study of the "primary incubation zones" previously cited discloses the fact that from the cultural standpoint they are ideal locations for the development and increase in virulence of anaërobes. Here the amount of free oxygen is greatly restricted, and as a rule, reducing substances are present. The many crevices, pockets, hidden localities and regions of food-impaction form the background for the growth of bacteria and complicate the pathological pictures exhibited in the mouth.

From the writer's observations the most important of the "primary incubation zones" in the development of necrotic gingivitis is the gingival flap associated with a partly or imperfectly erupted third mandibular molar. Here physical and chemical conditions that tend to anaërobiosis are usually constant.

The palatal margins of interproximal gingivae of upper central incisors are frequently the site of early lesions of necrotic gingivitis. Associated deep gingival crevices or pre-existing periodontal pockets furnish the basis for the establishment of the necessary "primary incubation zones". With gingival inflammation and swelling of the soft tissues, common to this region, deep thin pockets are formed which create, as in the case of the third mandibular molar, long capillary spaces containing fluid.

Capillary spaces wherever found or wherever produced, provided their base is in gingival tissue, may readily become "primary incubation zones". A common example of this is to be found where teeth are in unduly close apposition near the gingival line. This may come about through various malpositions, such as tilting of roots of adjacent teeth towards each other, or the rotation of teeth whereby the teeth fail to make interproximal contact in a normal manner. This latter condition is frequently seen where there has been drifting following extraction.

2. Secondary Incubation Zones

"Secondary incubation zones" may be defined as regions which are relatively unsuitable for the incubation and increase in virulence of the organisms and in which, from clinical observation, the primary planting of organisms of low virulence would not lead to disease. These areas become significant when they occur in a mouth wherein "primary incubation zones" have already become established and when the resistance of the tissue elements has been lowered by various factors. In passing, it might be noted that these secondary zones frequently present the outstanding evidence of disease in necrotic gingivitis in contra-distinction to the primary zones in which the symptoms are often less noticeable or even less acute.

The part played by traumatogenic occlusion in the production of "secondary incubation zones" is extremely important. The effect is that of a depression of tissue-tone which results in a lowering of resistance. Examples of this relationship are common in necrotic gingivitis, that is to say, cases are observed in which the only lesions to be found, with the exception of those located in the "primary" zones, occur where a definite traumatogenic occlusion can be demonstrated.

Another phase of this relationship is frequently to be seen in cases of recurrent necrotic gingivitis. Here the zone of incubation is very commonly found to be the site of an unrelieved traumatogenic occlusion. The importance of this finding in the treatment of both primary and recurrent cases amply warrants emphasis and even reiteration.

F. Histo-Pathology of Necrotic Gingivitis

The lesion consists essentially of an acute inflammatory process accompanied by necrosis arising on the surface and progressing more deeply with the advance of the disease.

The inflammatory process bordering the gingival surface consists of an acute polymorphonuclear infiltration scattered through the interstices of the covering epithelium and in the supporting tissues of the marginal gingiva. This acute inflammatory reaction is present both on the outer free surface as well as on its inner crevice surface. In association with this acute inflammatory reaction a necrosis and desquamation of the superficial epithelium soon follows accompanied by a marked inflammatory exudate made up largely of fibrin which incorporates the cells of the inflammatory exudate as well as the necrotic cells of the antecedent epithelium. Thus in relation to the acute inflammatory process attacking the gingival margin an ulcerating area covered by a firmly adherent pseudomembrane makes its appearance. This membrane is firmly implanted upon the ulcerating area, and sends projections of fibrin deeply into the neighbouring tissues forming the base of the ulcer.

Accompanying this ulcerating inflammatory process, bacteria are found to be incorporated in the deposit or pseudomembrane covering the surface. It is to be noted that although the inflammatory reaction is one in which the exudate is of the acute polymorphonuclear variety, there is no suppurating process with local abscess formation in the gingival tissues.

The acute inflammatory process is largely localized about the ulcerating lesion at the surface, although the influence of the bacterial attack has led to a considerable inflammatory infiltration of the deeper tissues. Furthermore, it is to be noted that the lesion is relatively localized, and does not tend to extend further than a few millimeters from the crest of the gingival margin. Nevertheless the necrosing process causes a complete destruction of the free margin, there apparently being no immunity of the tissues of either the inner or outer surface.

The inflammatory reaction which is found in the gingival tissues remote from the necrotic and ulcerating process loses the quality of an acute polymorphonuclear reaction and shows the presence mainly of an infiltration by plasma cells. There is no sharp line of demarcation between the area involved in inflammation and the healthy tissues.

There is a distinct difference between the lesion as here observed in necrotic gingivitis, and gangrenous stomatitis. In the former as we have just pointed out the line of demarcation between the advancing border of the inflammatory process and the healthy tissues is not sharply defined, while in the gangrenous stomatitis the advancing border forms a very sharply demarcated zone in which bacteria and inflammatory exudate are recognized. In this latter type of lesion an inflammatory exudate does not extend distinctly beyond the necrosing or gangrenous process.

G. Treatment

The case-management of necrotic gingivitis, as a rule, is divided into three main steps, as follows:

- (a) Treatment of the acute phase.
- (b) Elimination of the "secondary incubation zones".
- (c) Elimination of the "primary incubation zones".

Where the gingivitis is of the sub-acute type, the management of the average case resolves itself into the eradication of the incubation zones.

- (a) Treatment of the Acute Phase---This comprises the following procedures:

(1) Topical Application of Medicaments

I. Adams' Treatment

In necrotic gingivitis the local medicinal treatment widely used by Canadian dentists for years is the Tincture of Iodine-Silver Nitrate Combination. In severe cases and where the technique outlined is strictly followed, the results as a rule are spectacular. Its peculiar affinity for necrotic tissue, the rapid shedding of the ulcerous conglomerate with restoration of a vigorous epithelial covering, and the almost immediate relief from soreness and pain are attributes which make this treatment a specific for this type of infection.

The solutions used in this combination-treatment are:

- a) Churchill's Tincture of Iodine.

The formula is as follows:

Iodine	16.5 grams
Potassium Iodide	3.3 grams
Alcohol (70%)	100 c.c.

All the soft tissue surfaces are freely endowed with tiny crevices and microscopic interspaces in which are present, in necrotic gingivitis, large numbers of microorganisms. Into these minute openings the iodine solution must necessarily penetrate. Churchill's tincture of iodine, because of its low surface tension, rapidly flows to the gingival crevices of teeth adjacent to the point of application and readily seeps into all the microscopic irregularities.

- b) 35% aqueous solution of Silver Nitrate.

Steps in the topical application of the Tincture of Iodine-Silver Nitrate Combination:

- a) Place on the bracket-table two glass containers (inverted glass castor-cups serve the purpose very well) and, governed by the area of soft tissue to be treated, place on one of them 5 to 10 pellets of cotton, match-head in size, and saturated with Churchill's tincture of iodine. On the other, 5 to 10 pellets of cotton, slightly larger than the head of a match, and saturated with 35% aqueous solution of silver nitrate are placed. The making of cotton-pellets and dipping into medicine-bottles by the operator after the treatment has begun are impedient procedures that tend to make the consecutive application of the medicaments more difficult.
- b) Next isolate the affected area with cotton rolls, and with sterile cotton pellets gently dry the tissues, being careful not to induce bleeding. (It may be pointed out here that Adams did not advocate the drying of the tissues before the application of the medicaments.)
- c) One of the cotton pellets saturated with Churchill's tincture of iodine is taken in the pliers and lightly touched to the amelogingival border. With slight compression of the pellet, the liquid flows rapidly because of its low surface tension over the ulcerated tissues, along the gingival crests, into the gingival crevices and between the teeth. Care must be taken that the tincture is not allowed to flow over the alveolar or other oral mucosa, as it occasionally has an escharotic action. The use of

small pellets of cotton, saturated but without great excess of tincture, the gentle contacting with the tissues, preceded by their careful drying, tend to limit the flow of the solution to the parts affected.

Where there has been great loss of tissue between the teeth, frequently with marked cupping-out of the structures, and close apposition of the roots, difficulty is occasionally experienced in the placing of the tincture at the base of this inter-dental space. This is easily overcome by the use of the chip-blower. Having first removed the saliva and detritus and deposited the tincture, with a light current of air the tincture is gently blown to the base of the space, to be followed at once with the solution of silver nitrate.

- d) A pellet saturated with the 35% solution of silver nitrate is immediately carried in the pliers to the regions holding the Churchill's tincture and gently compressed. The tiny crevices and fissures again act as sluice-ways for the silver nitrate solution, which combines with the iodine tincture resulting in the formation of a bright whitish-yellow precipitate in the necrotic and ulcerated tissue. The tooth is left stainless when aqua ammonia is applied to it.

The chemical reaction between the two solutions results in the precipitation of the insoluble iodide of silver which seems to form within the necrotic tissue. This whitish film turns dark in the light and in thirty-six hours as a rule, is cast off. It seldom leaves behind a bleeding surface, all ulcerations except the large ones being covered as a rule with new epithelium. Furthermore, the rapid reduction of the attendant gingival inflammation is a particularly striking feature.

In effect the treatment accomplishes the same result as is accomplished in the tannic acid treatment of burns. The penetration of the antiseptics not only kills the organisms but also the superficial tissue through coagulation of the tissue proteins, and just as in the tannic acid treatment, this coagulation appears to render the dead tissue resistant to the action of the bacteria. We have in fact created a perfectly-fitting sterile dressing under which a healthy epithelial growth may occur, and this indeed invariably takes place.

When the areas selected for treatment have been attended to, the writer advises the thorough irrigation of the mouth with plain warm water. At least four glasses of water are given to the patient for repeated rinsings to insure the removal of excess silver nitrate.

II. Dental Mapharsen

This product has proved to be, in the writer's experience, the most effective of the arsenical preparations in the more severe forms of necrotic gingivitis. Where the lesions are confined to the gingival margin, favourable results have been uniformly and promptly produced. Mapharsen is a trivalent arsenical, the hemialcoholate of 3-amino-4-hydroxyphenylarsine oxide hydrochloride. It is supplied for clinical use in powder form, mixed with alkali and sucrose, an aqueous solution being practically neutral and isotonic with the blood. In necrotic gingivitis,

mapharsen is used topically in powder form or in solution. When applied in powder form directly to the lesions it is most efficacious. It may also be used after dissolving in water or mixing in glycerine. The writer uses a small, silver, shovel-shaped instrument (Platt) to deposit the mapharsen on the surface of the lesions and under the gingival margins. This applicator is first dipped in water to moisten the surface, then dipped into the powder which is then carried to the tissues. The special shape of the instrument facilitates the introduction of the drug to subgingival regions. The contents of one 60 mg. ampoule are used in each treatment. Severe cases are treated daily, the milder cases every second day.

III. Lactis-ora

This product has yielded very satisfactory results in the treatment of the milder forms of necrotic gingivitis, Lactis-ora is a dark metallic-appearing solution, the active ingredients of which are phenylacetamide, cinchotannic acid, oleum sassafras and chromium trioxide. It is made in two strengths, concentrated and ordinary, the first for professional use, and the second to be used by the patient as a home treatment.

IV. Paraffin packing

The use of paraffin packing is recommended for the relief of pain associated with necrotic lesions of the gingival margin. Gottlieb¹⁷ has pointed out that the pain in acute necrotic gingivitis is principally due to the irritating effect of saliva on the surface of these lesions, and that when they are covered with packing the pain is quickly relieved. The lesions also are repaired with greater rapidity.

(2) Instruction in home mouth-washing

The writer has found the use of the following mouth-wash to be helpful in cases presenting lesions of necrotic gingivitis. It is used, as a rule, three or four times a day for the first three or four days, then less frequently. The patient is given the following prescription:

Potassii nitrat	℥ j	(drachm)
Boracis	℥ ij	(drachms)
Potassii chlorat	℥ iv	(drachms)
Tr. arnicæ	℥ iv	(drachms)
Aq. rosae ad	℥ xii	(ounces)

M.

Sig: To be used as a mouth-wash with an equal volume of warm water as directed.

It is emphasized that during the acute phase of necrotic gingivitis, extractions, scaling or other oral surgical procedures are contra-indicated.

With the subsidence of the acute symptoms scaling may be instituted. Extractions, however, should be postponed until all signs of the disease have disappeared.

Rosenthal¹⁸ warns against the use of general anaesthetics even in mild cases of Vincent's infection.

Before dismissing the patient at the first visit, instructions are given regarding the use of separate dishes, glasses and eating utensils. These should be boiled after using.

(b) Elimination of the "Secondary Incubation Zones"

It has been stated previously that frequently the lesions developed at the sites of the "secondary incubation zones" form the background of the typical clinical picture of necrotic gingivitis and stomatitis. Through the various factors which have permitted the colonization of the microorganisms in these relatively unfavourable regions, a localization at definite sites, of the characteristic lesions occurs, and which constitutes a salient feature of the disease. These factors include traumatogenic occlusion, gingival irritants and pocket formation. Elimination of these factors necessitates an appreciation of modern periodontal case-management.

(c) Elimination of the "Primary Incubation Zones"

The case-management of necrotic gingivitis is obviously incomplete without the eradication of those regions known as the "primary incubation zones". It will be recalled that here through exceptionally favourable cultural conditions, the microorganisms have proliferated rapidly and increased in virulence. From these zones, the bacteria are transferred to sites of new colonization.

Recurrent attacks of necrotic gingivitis, after intermissions and frequently following treatment, are common. Almost invariably, this recrudescence may be explained by the presence in the mouth of an active and unrecognized primary incubation zone. Therefore, as a prophylactic measure against reinfection, the elimination of this zone is of extreme importance.

According to the foregoing classification, the primary zone of greatest significance is the gingival flap associated with partly or imperfectly erupted lower third molars. In many examples, the eradication of the accompanying pocket can best be accomplished through the removal of the partly erupted third molar. Due to the relation, frequently abnormal, of the tooth to its tissues, excision of the overlying gingival tissue fails to render the pocket depth into one of physiologic shallowness.

Certain examples are present where with excision of the overlying gingival flap and consequent tissue contraction, the pocket is made shallow enough to permit the establishment of hygienic conditions through careful brushing.

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STUDY NO. 12

A PERIODONTAL ASPECT OF CARIES IMMUNITY

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Outline - A. General Manifestations of Immunity to Dental Caries

B. Local Manifestations of Immunity

- (a) All teeth are not equally prone to caries
- (b) All tooth surfaces are not equally susceptible

C. The Problem of Certain Factors Concerned in Caries Immunity

- (a) Periodontal conditions that seem to protect against caries
- (b) The suggestion of another protective mechanism on the tooth surface

D. Bibliography

A PERIODONTAL ASPECT OF CARIES IMMUNITY

A. General Manifestations of Immunity to Dental Caries

That the external and internal coatings of the body furnish a mechanical defense against bacterial invasion is well known. The unbroken skin forms an effective protection against bacteria, and the mucous membrane while not the equal of the skin, is nevertheless a fairly resistant agent. This is especially true of the unbroken mucous membrane of the oral cavity.

The enamel, a cuticular production of epithelial origin is the main protection of the dentine and dental pulp. It in turn is protected by two membranes or cuticles. The first or inner cuticle which is the final product of the ameloblasts, when calcified is indistinguishable from the enamel proper. According to Orban¹ it is no more resistant to acids or alkalis than the enamel itself. Maximow and Bloom² have stated that this membrane is "somewhat more resistant to acid than the rest of the enamel as it contains less calcium." According to Bennett³ Thewlis working in the National Physical Laboratory of London has demonstrated "that there is a radiographically dense skin of enamel over the outer surface." The second or outer cuticle is a keratinous membrane which ordinarily is largely worn off in adult life. Orban has stated that "frequently the secondary cuticle is not formed."

In dental caries, certain conditions prevail which apparently favor the colonization of acid-forming bacteria on the tooth surface and the production of acid under circumstances that enable it to initiate the caries process. The factors that tend to inhibit the establishment of these conditions necessary to the inception of caries constitute what in a broad sense may be termed immunity to this disease.

In an examination of the teeth in British Neolithic skeletons discovered at Coldrum, Kent, Cameron⁴ was unable to detect a single case of caries. Here is exemplified a type of natural immunity that may be termed absolute. And, individuals are occasionally seen in practice, who, living under modern conditions present the same state of immunity. Nowhere on the surfaces of the teeth are the microorganisms associated with caries permitted to maintain themselves under conditions suitable for the production of their deleterious effects on the enamel, nor do acids from other sources attack the enamel surfaces.

In other groups of people and individuals, the natural immunity is relative, that is to say it has limitations. In a previous paper, it was stated that the skeletal remains of Neanderthal man showed no evidence of dental caries.⁵ It is known, however, that man was afflicted with caries in very early times. Recently, on the authority of Baegge of Berlin, it is stated that dental caries occurred here and there in a scattered manner as early as the Mesolithic age. Increasing in frequency, at the termination of the Neolithic age, it is said to have reached modern proportions.

It must be pointed out, nevertheless, that wide variations in their susceptibility to dental caries have existed and still exist among different primitive peoples. According to the findings of Mummery⁶ in the Eskimos, one case was found in 69 skulls. In 1939, Williams*, on the Eastern Arctic Expedition, in an examination of Eskimos living close to their natural conditions, found an absolute immunity to dental caries. White flour, white sugar, baking powder and tea made up not more than five to ten per cent. of their total diet. In 51 nomad Eskimos in small groups available at the trading posts, 1418 teeth were examined and no carious cavities found. Mummery found caries in 24 out of 60 skulls from long barrows in Wiltshire, the burials in all probability belonging to Neolithic times in England. And according to Keith⁷ the teeth of Rhodesian man (one specimen) discovered in the Broken Hill mine, Northern Rhodesia, in a filled-up cave, were ravaged by caries.

The investigations of Osborn and Noriskin⁸ show that the South African Bantu is relatively immune to caries, while living in the primitive manner of the kraal native. In an examination of pre-historic Bantu skulls, 40 in number, out of 749 teeth, 31 were carious. These investigators have estimated the percentage of primitive kraal natives with caries as ranging from twenty to thirty per cent. They regard this as a small matter as compared to the great susceptibility to caries of the teeth of the Bantu under civilized conditions.

It is commonly understood that the teeth of the Eskimo show a marked susceptibility to dental caries under environmental conditions common to the trading post. Williams, in an examination of 10 Eskimos living at the posts either as employees or children of employees of the white men, found 39 carious cavities in 262 teeth. White flour, white sugar, baking powder and tea comprised about twenty-five to thirty per cent. of their diet.

The general observations of the dental profession would seem to confirm the viewpoint that in certain mouths hitherto relatively immune to caries, there is manifested a state of marked susceptibility and which is frequently of a more or less temporary nature. For example, it is commonly believed that in pregnancy there is, as a rule, a greatly increased predisposition to caries.

It is of great interest to note that Graham⁹ in a study of the reaction of the salivas from the different salivary glands proper in 31 pregnant women, found 81 per cent. of the glands secreting saliva at the mouth of the ducts acid in reaction. In a married control group of 31 women, the percentage of glands secreting acid saliva dropped to 50, while in an unmarried control group of 31, only 39 per cent. of the glands showed saliva acid in reaction.

It has been stated also that following continued fevers, where the saliva has been noticeably sparse in amount for some time, a period of greatly increased susceptibility to caries frequently follows. In mouths that have manifested a marked tendency to caries, it has been observed also that changes take place which bring about a state of relatively increased immunity.

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B. Local Manifestations of Immunity

(a) All teeth are not equally prone to caries.

According to the figures of Scheff¹⁰ out of 1000 carious teeth, 647 were found in the upper jaw and 353 in the lower jaw. Also, in an analysis made by Wallis and Pare¹¹ of the relative frequency of caries in over 10,000 extractions, it was revealed that the upper teeth, with the exception of the second and third molars, as compared to the lowers, showed definitely a greater disposition to caries.

It is a matter of common observation, as has been pointed out by the writer,¹² that in a great many mouths the lower anterior teeth exhibit a marked immunity to caries, cervical and proximal. Certain environmental conditions peculiar to this region evidently prevent, in some manner, the initiation of caries on these tooth surfaces.

It has been shown previously that frequently the lower anterior region of the oral vestibule is under the dominance of alkaline or neutral saliva, in spite of the fact that the parotid saliva may be acid in reaction. This is due to the existence of an alkaline reaction in the mandibular saliva which influences the prevailing acid reaction of the vestibule in the lower anterior region.

(b) All tooth surfaces are not equally susceptible.

It has often been stated that the general regions on the tooth surfaces that are prone to caries are three in number, viz: in pits and fissures; on proximal surfaces under the contact point; and on lateral and buccal surfaces near the gingival line.

The lingual surfaces of the teeth are not commonly attacked by cervical caries, which in the main, is a lesion of the oral vestibule. Cervical caries rarely invades the gingival crevice. The caries process seldom, if ever, begins on the tooth surface under the gingival margin.

It has been stated by some writers that proximal and cervical caries occurs on surfaces that fail to be scoured by the passing over of food in these localities. This is apparently true under certain conditions when a state of general susceptibility to caries exists in the individual. However, it must be pointed out that many cases are to be observed where caries fails to be initiated on poorly cleansed proximal and cervical tooth surfaces, i.e. surfaces that show the presence of a soft, adhesive material by revealing stain or other test.

C. The Problem of Certain Factors Concerned in Caries Immunity

(a) Periodontal conditions that seem to protect against caries.

Cases are frequently encountered where the tooth surfaces are in an unclean condition and no sign of the caries process is to be observed with the magnifying glass when the surfaces are cleansed. The portions of the enamel surface that tend to be unscoured, e.g. the gingival third of the labial and buccal surfaces, and occasionally

the remaining surface as well, are covered with a slippery, glutinous coating. Good examples of this condition are to be observed on the disuse side in cases where there is masticatory function on one side only. This viscous deposit contains desquamated epithelial cells, leucocytes, mucus, and other protein bodies. As Wallace¹³ has pointed out, these are continually being shed into the buccal cavity. According to the writer's tests, these deposits are almost invariably alkaline in reaction. This coating, as would be expected, is commonly found in cases of chronic gingival inflammation and periodontal disease. The albuminous material forming this layer apparently constitutes a suitable medium for certain bacteria that are not acid-forming, hence the freedom from decalcification of the enamel surface ^{as revealed} on the removal of the debris. That this coating is protective in nature is attested to by the fact that when it is artificially kept brushed away, caries, in many cases, soon makes its appearance. The writer feels sure that every periodontist has observed this phenomenon. Noyes¹⁴ has pointed out that "caries shows the greatest intensity in comparatively clean mouths", and Wallace¹⁵ has observed that "the ill-cared-for and dirty mouths of hospital patients may be remarkably free from dental caries."

Examples of decalcifications of the surface enamel are encountered which at the time of examination are still in the early stages. At the same time, they are of long standing. These decalcified defects are found on clinical examination to be covered with a thin, slippery coating or film. This coating is mainly confined to these whitened areas and is found to be alkaline in reaction. On microscopic examination of smears made from these regions, this glutinous material was found to be largely comprised of leucocytes and epithelial cells. The writer looks upon the presence of these films as constituting a "passive" phase in the caries process. The "active" phase of the lesions of incipient caries is essentially one of acid production on the enamel surface through the action of microorganisms on carbohydrate material. The acids are so protected from the saliva that sufficient intensity is achieved to produce decalcification. For the "active" phase to be continuous, it is plain that the carbohydrate content of the plaque or debris must be restored almost constantly. Otherwise on depletion of the carbohydrate supply, the acidogenic bacteria find the changed medium unsuitable for their cultural conditions and fail to proliferate.

It has been observed that caries, once established, is usually slow in progression on the occlusal surfaces of non-functioning teeth. This has been ascribed to the failure of periodic replenishment of the carbohydrate content of the crevices and grooves, as stagnation is present in these regions. That the grooves of non-functioning occlusal surfaces frequently become filled with a hard calcified material is well known. Apparently the stagnant debris, through development of an alkaline reaction and with seepage of saliva, becomes the matrix for precipitated calcium salts, and calculus is thereby formed. This is opposed to the caries process (Miller).

Black¹⁶ called attention, over fifty years ago, to the clinical observation that the presence of pus is preventive of caries. He has stated that decay in a cavity ceases if pus is continually discharged into it, e.g: a chronic alveolar abscess discharging through the pulp canal. Root surfaces that are exposed, either through eruption or recession, or where the crowns have been lost, seldom are attacked by caries in an environment of gingival pus production.

It also has been referred to previously that the tooth surfaces in the gingival crevices show a pronounced freedom from caries attack. In hundreds of tests, it has been found that the gingival crevice is usually slightly alkaline in reaction.

It is to be noted that in these protective conditions--the glutinous coating, the presence of pus, and the crevice contents--there appears to be one common factor, viz: alkalinity of reaction. While this factor may not be the sole, or even the principal agency of protection against caries in these conditions, nevertheless it would appear to play a part.

(b) The suggestion of another protective mechanism on the tooth surface

(1) Enamel protection in an acid saliva-bread mixture*

When a tooth is suspended in a saliva-bread mixture, incubated for three or four days at 37° C. no evidence of decalcification of the enamel is manifested. On the other hand, a tooth suspended in a fermenting bread mixture to which no saliva has been added, and under the same conditions of time and temperature, shows definite evidence of decalcification of the enamel surface. The reaction of the mixture in each case becomes fairly acid, i.e. about pH 4.5 in a few hours. It seems apparent that in the first instance where the saliva is present some protective factor is operative and which is absent in the second case. This is not to say, however, that the value of this protective mechanism is equal in all salivas.

(2) When a tooth is suspended in a weak solution of HCl to which a small amount of sodium oleate has been added, the pH being approximately 4.5, the enamel surface shows no decalcification for some days. A tooth suspended in a weak solution of HCl, with a reaction of approximately pH 4.5 shows evidence on the enamel surface of the process of decalcification in a few hours.*

It was pointed out in a recent bulletin¹⁷ that teeth suspended in a buffered solution pH 5.0, without the addition of sodium oleate showed marked decalcification of the enamel surface in 24 hours. Here also it was shown that the addition of sodium oleate to the same buffered solution protected the enamel surface from attack for several days. As the soaps of the higher fatty acids can not exist in solutions under pH 9.0, it seemed probable that the sodium oleate dissociated, leaving a film of oleic acid on the teeth in question. Subsequent

*These experiments were carried out by Mr A. F. Fenton, Research Technician, Department of Periodontology, University of Toronto.

examination showed this to be the case.

(3) Teeth dipped in oleic acid, and suspended in a weak HCl solution and a buffered solution pH 5.0, were then found to exhibit the same protection against decalcification as in the former experiments where sodium oleate was used.*

These findings suggested the possibility that the production, in some manner, of higher fatty acid films or emulsions on the enamel surfaces of the teeth suspended in the saliva-bread mixture, accounted for the protection of these surfaces against decalcification. It is well known that starch is comprised mainly of two distinct fractions termed amylose and amylopectin. It was of great interest to learn that amylopectin shows a tendency to esterification, and Ruhland and Wolf¹⁸ point out that "in the seeds of cereals, it is esterified with palmitic, oleic and linolic acids." Taylor and Sherman¹⁹ were able to split up this ester by the action of lipase-free amylase, as well as by acids and bases. They found, for example, the fatty acid content of whole corn starch to be 0.73 per cent. The amylopectin fraction of corn starch yielded approximately 4 per cent of fatty acids. It therefore seems reasonable to assume that, in the saliva-bread mixture previously described, the salivary liquefying amylase liberated the higher fatty acids of the cereal starch, and that this mechanism occurs in the oral cavity.

From experiments carried out in our laboratory, the evidence points to wide variations in the amounts of higher fatty acids liberated by equal amounts of different salivas, and of the same saliva at different times. And the amount of higher fatty acids produced appear to be in direct ratio to the activity of the liquefying amylase described in a previous bulletin²⁰. A deficiency of this protective material on the tooth surfaces would seem to be related to the extent of the caries attack in an individual mouth. In other words, adequate production of fatty acid films or emulsions on the teeth would seem to give protection more or less generally, and to limit the process of caries, when initiated, to regions dominated by peculiar local conditions.

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